In the Claims

For the Examiner's convenience, all of the pending claims, whether or not amended, are set forth below.

Please amend the following claims:

1. (Amended) A method for selectively modulating a Th2-type response within a population of activated CD4+ T cells, comprising contacting the population of activated CD4+ T cells with an agent which modulates a B7-2-induced signal in the population of activated CD4+ T cells, such that the Th2-type response is modulated.

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2. (Amended) The method of claim 1, wherein the Th2-type response is stimulated by contacting the population of activated CD4+ T cells with an agent which stimulates a B7-2-induced signal.

3. (Amended) The method of claim 2, wherein the agent which stimulates a B7-2-induced signal in the population of activated CD4+ T cells is a stimulatory form of B7-2.

4. (Amended) The method of claim 3, wherein the stimulatory form of B7-2 is a form of B7-2 which is attached to a solid phase support.

5. The method of claim 4, wherein the solid phase support is a surface of a cell.

6. The method of claim 3, wherein the stimulatory form of B7-2 is a soluble form of B7-2.

7. The method of claim 6, wherein the soluble form of B7-2 is a fusion protein.

8. The method of claim 7, wherein the B7-2 fusion protein is a B7-2-immunoglobulin fusion protein.

9. (Amended) The method of claim 1, wherein the Th2-type response is inhibited by contacting the population of activated CD4+ T cells with an agent which inhibits a B7-2-induced signal.

10. (Amended) The method of claim 9, wherein the agent which inhibits a B7-2-induced signal in the population of activated CD4+ T cells is an agent which inhibits an interaction between B7-2 and a B7-2 ligand on the CD4+ T cells.

11. The method of claim 10, wherein the agent that inhibits an interaction between B7-2 and a B7-2 ligand is an anti-B7-2 antibody.

12. (Amended) [A] The method of claim 1, wherein the population of activated CD4+ T cells is activated by [for selectively modulating a Th2-type response within a population of activated CD4+ T cells, comprising] contacting CD4+ T cells with [a first] an agent which provides a primary activation signal to the CD4+ T cells [and a second agent which modulates a B7-2-induced signal in the CD4+ T cells, such that the Th2-type response is modulated].

13. (Amended) The method of claim 12, wherein the Th2-type response is stimulated by contacting the population of activated CD4+ T cells with [a first agent which provides

a primary activation signal to the T cells and a second] an agent which stimulates a B7-2-induced signal in the CD4+ T cells[, such that the Th2-type response is stimulated].

14. (Amended) The method of claim 13, wherein the [second] agent which stimulates a B7-2 induced signal is a stimulatory form of B7-2.

15. (Amended) The method of claim 14, wherein the stimulatory form of B7-2 is a form of B7-2 which is attached to a solid phase support.

16. The method of claim 15, wherein the solid phase support is a surface of a cell.

17. The method of claim 14, wherein the stimulatory form of B7-2 is a soluble form of B7-2.

18. The method of claim 17, wherein the soluble form of B7-2 is a fusion protein.

19. The method of claim 18, wherein the B7-2 fusion protein is a B7-2-immunoglobulin fusion protein.

20. (Amended) The method of claim 12, wherein the [first agent] agent which provides a primary activation signal to the CD4+ T cells is an anti-CD3 antibody.

21. (Amended) The method of claim 12, wherein the [first agent] agent which provides a primary activation signal to the CD4+ T cells is an antigen presented by an antigen presenting cell.

22. (Amended) The method of claim 12, wherein the [first agent] agent which provides a primary activation signal to the CD4+ T cells is a protein kinase C activator and a calcium ionophore.

23. (Amended) [A] The method of claim 1, wherein the contacting is in [for treating] a subject having a condition that can be ameliorated by modulating a Th2-type response, [in the subject, comprising administering to the subject an agent which modulates a B7-2-induced signal in the CD4+ T cells], such that a Th2-type response is modulated in the subject to thereby ameliorate the condition in the subject.

24. (Amended) The method of claim 23, wherein the agent stimulates a B7-2-induced signal in the population of activated CD4+ T cells [such that a Th2-type response in the subject is stimulated to thereby ameliorate the condition].

25. (Amended) The method of claim 24, wherein the agent which stimulates a B7-2-induced signal in the population of activated CD4+ T cells is a stimulatory form of B7-2.

26. The method of claim 25, wherein the stimulatory form of B7-2 is a form of B7-2 which is attached to a solid phase support.

27. The method of claim 26, wherein the solid phase support is a surface of a cell.

28. The method of claim 25, wherein the stimulatory form of B7-2 is a soluble form of B7-2.

29. The method of claim 28, wherein the soluble form of B7-2 is a fusion protein.

30. The method of claim 29, wherein the B7-2 fusion protein is a B7-2-immunoglobulin fusion protein.

31. The method of claim 24, wherein the condition is an autoimmune disease.

32. The method of claim 31, wherein the autoimmune disease is rheumatoid arthritis.

33. The method of claim 31, wherein the autoimmune disease is multiple sclerosis.

34. The method of claim 31, wherein the autoimmune disease is type I diabetes.

35. The method of claim 24, wherein the condition is an infection with an infectious agent.

36. The method of claim 35, wherein the infectious agent is a parasite.

37. (Amended) The method of claim 23, wherein the agent inhibits a B7-2-induced signal in the population of activated CD4+ T cells such that a Th2-type response in the subject is inhibited to thereby ameliorate the condition.

38. (Amended) The method of claim 37, wherein the agent which inhibits a B7-2-induced signal in the population of activated CD4+ T cells is an agent which inhibits an interaction between B7-2 and a B7-2 ligand on the CD4+ T cells.

39. The method of claim 38, wherein the agent which inhibits an interaction between B7-2 and a B7-2 ligand is an anti-B7-2 antibody.

40. The method of claim 37, wherein the condition is an allergy.

41. The method of claim 37, wherein the condition is an infection with an infectious agent.

42. (Amended) [A] The method of claim 1, wherein the contacting is *ex vivo* and the method further comprises [for treating a subject having a condition that can be ameliorated by modulating a Th2-type response in T cells of the subject, comprising

(a) obtaining a population of cells comprising CD4+ T cells from the subject;

(b) contacting the CD4+ T cells with an agent which modulates a B7-2-induced signal in the CD4+ T cells such that a Th2 response is selectively modulated within the population of CD4+ T cells; and

(c) readministering] administering the CD4+ T cells to [the] a subject having a condition that can be ameliorated by modulating a Th2-type response in T cells of the subject.

43. The method of claim 42, wherein the CD4+ T cells are contacted with an agent that stimulates a B7-2-induced signal in the CD4+ T cells such that a Th2 response is selectively stimulated.

44. (Amended) The method of claim 43, wherein the population of activated CD4+ T cells is activated [T cells] with the agent that stimulates a B7-2-induced signal in the

CD4+ T cells together with [a second] an agent that stimulates a primary activation signal in the CD4+ T cells.

45. The method of claim 43, wherein the agent which stimulates a B7-2-induced signal in the CD4+ T cells is a stimulatory form of B7-2.

46. The method of claim 45, wherein the stimulatory form of B7-2 is a form of B7-2 which is attached to a solid phase support.

47. The method of claim 46, wherein the solid phase support is a surface of a cell.

48. The method of claim 45, wherein the stimulatory form of B7-2 is a soluble form of B7-2.

49. The method of claim 48, wherein the soluble form of B7-2 is a fusion protein.

50. The method of claim 49, wherein the B7-2 fusion protein is a B7-2-immunoglobulin fusion protein.

51. The method of claim 42, wherein the CD4+ T cells are contacted with an agent which inhibits a B7-2-induced signal in the CD4+ T cells such that a Th2 response is selectively inhibited.

52. The method of claim 51, wherein the agent which inhibits a B7-2-induced signal in the CD4+ T cells is an agent which inhibits an interaction between B7-2 and a B7-2 ligand on the T cells.

53. The method of claim 52, wherein the agent inhibits an interaction between B7-2 and a B7-2 ligand is an anti-B7-2 antibody.

54. A packaged form of an agent which stimulates a B7-2-induced signal in a population of CD4+ T cells to selectively stimulate a Th2-type response in the population of CD4+ T cells packaged with instructions for using the agent to selectively stimulate a Th2-type response in a population of CD4+ T cells.

55. The packaged form of claim 54, wherein the agent which stimulates a B7-2-induced signal in a population of CD4+ T cells is a stimulatory form of B7-2.

56. A packaged form of an agent which inhibits a Th2-type response in a population of CD4+ T cells by inhibiting a B7-2-induced signal in the CD4+ T cells packaged with instructions for using the agent to selectively inhibit a Th2-type response in a population of CD4+ T cells.

57. The packaged form of claim 57, wherein the agent which inhibits a Th2-type response in a population of CD4+ T cells is an antibody to B7-2.

58. The packaged form of claim 54 wherein the agent is a therapeutic composition and the instructions are for therapeutic administration.

59. The packaged form of claim 56 wherein the agent is a therapeutic composition and the instructions are for therapeutic administration.